serious of all the disadvantages. There are a number of useful suggestions in the paper, but they do not have very ready usage here.

5. *Lunacy and the Law.*—T. O. Wood discusses several features in the English Lunacy Law, which are of secondary interest to those practicing under different regulations.

6. *Note on a New Case Book Form.*—This is a short note on the method of keeping clinical histories and has no particular points one way or another.

7. *Hallucinations.*—The author here gives the histories of thirteen patients suffering from different forms of hallucinations. The paper is too detailed to admit of ready abstraction, but it contains many interesting suggestions, bearing more particularly upon the theory of psycho-motor verbal hallucinations, whereas he does not discuss any of this type. Those that are given show interesting connections with the forms described by Seglas.

8. *Clinical Notes and Cases.*—This number also contains Notes on some Pathological Cases, by M. J. Nolan, the swallowing of foreign bodies, such as nails, bristles, etc., by dements; a case of suppurative cholangitis; melancholia with universal acute eczema; senile melancholia associated with aneurisms of the aorta and rupture, and also a few Cases of Abdominal Surgery in the Insane, by Robert Jones; A Case of Hebephrenia, by W. R. Dawson; a Case of Thoracic Aneurism in a Secondary Dement, Simulating Mediastinal Growth, by Robt. Pugh; and Status Epilepticus Complicated with Scarlet Fever, by G. W. Greene. JELLIFFE.

BRAIN

(Vol. 25, 1902, Winter, No. 4.)

1. Some Observations on the Primary Degeneration of the Motor Tract. F. W. MOTT and A. F. TREDGOLD.
2. On the Pallio-tectal or Cortico-mesencephalic System of Fibers. CHARLES E. BEEVOR and SIR VICTOR HORSLEY.
3. A Case of Arrested Development of the Cerebellum and its Peduncles; with Spina-Bifida and Other Developmental Peculiarities in the Cord. W. B. WARRINGTON and K. MONSARRAT.
4. Toxic Degeneration of the Lower Neurones, Simulating Periphery Neuritis. STANLEY BARNES.
5. On Certain Blood Changes in Idiopathic Epilepsy. ROBERT PUGH.

1. *Primary Degeneration of the Motor Tract.* The authors describe the clinical course and microscopical appearances in four cases: two amyotrophic lateral sclerosis, one of progressive muscular atrophy and one of chronic rheumatoid arthritis with muscular atrophy. They say that degeneration of the motor tract is the anatomical lesion present in severe diseases having distinct clinical features. Thus it occurs in Friedreich's disease, in combined lateral and posterior sclerosis and in general paralysis, but in these other neurone systems are also affected. Degeneration may also result from local lesions as in the anterior poliomyelitis, polioencephalitis, embolus, etc. And lastly, cases exist in which not a degeneration but an arrest of the development of the motor tract occurs, apparently due to simple agenesis. It is with the primary degenerations, however, that occur in amyotrophic lateral sclerosis, progressive muscular atrophy, bulbar paralysis or primary spastic paraplegia that the paper deals.

Speaking of the nature of the morbid processes they believe that the changes in these cases are those of a sub-acute or chronic degeneration of a portion or the whole of the motor tract, that is of the upper segment alone, of the lower alone, or of both segments together. The cases they describe demonstrate that the change is a slow attack of individual neurones. The cells become shrunken and irregular in outline, the Nissl bodies gradually disappear, their space is taken by a large collection of yellow pigment, the nucleus becomes smaller and its outline displaced. Later it is displaced to the periphery of the cell and finally disappears. There is true degeneration in the nerve fibers and in the end organs. Neuroglia gradually takes the place of the atrophied nerve fibers causing a secondary sclerosis. The authors believe that this progressive degeneration is due to the fact that the neurone has reached the stage at which its inherited vitality is exhausted and that this presents a probable indication of presenile decay, but they present no evidence one way or another.

On the question of the pathological relations between the upper and lower motor segments they say that while there can be no doubt of the existence of trophic action between the upper and lower parts of the efferent tract, in some cases the presence of degeneration in one portion resulting from degeneration in the other seems to be rather the exception than the rule, and they consider that it is now well established that disease may involve either segment separately or both together, and if each segment of the motor tract is specially susceptible to the influence of particular poisons it seems legitimate to conclude that there may be different hereditary tendencies in the two tracts, in some persons there may be a premature tendency to decay in the upper portion. in others in the lower portion, and in still others throughout the entire motor system. Other neuronic involvement was present in some of the cases. These were considered secondary by the authors and were discussed under two heads, association fibers and efferent fibers.

2. *Pallio-tectal System of Fibers.*—An investigation of fibers composing the crus cerebri in a number of animals, 20 cats, 11 monkeys, 1 dog and 2 badgers. Local lesions in the cortex had been made by Horsley. While tracing the degerations which resulted from such lesions the authors especially followed out the connections which have for some years been known to exist between the cortex and the mid-brain. The Bush modifications of the Marchi method were used and serial sections studied. The results obtained are very detailed and the original should be consulted, but the general statement may here be made that as the research was founded chiefly on observations on the cat, representing the carnivora, and the macaque monkey, representing the primates, the results are to be understood as applicable only to the higher of these animals in which there is a well-developed non-excitable area (frontal) in front of the so-called motor or excitable cortex. The general scheme of the pallio-tectal system is stated by the authors to be as follows: From the frontal cortex no fibers can be traced to the mesencephalon; from the temporo-sphenoidal cortex very few fibers; while from the occipital lobe a fair number, and from the excitable cortex a large number of fibers can be followed to the corpora quadrigemina and mesencephalon. The course taken by the different group of fibers is then traced by the authors. We can not give the details in this place, but refer the reader to the original.

3. *Arrested Development in the Cerebellum.*—The authors describe in full the clinical history of a case of restricted development of the cerebellum, and then describe the macroscopical appearances of the brain and cord; the histology of the medulla, cerebellum, pons and membranes with the following results. A child, aged six weeks, presenting a lumbar spina bifida ruptured at or before birth, and now healed; hydrocephalus, paraplegia of both lower limbs. Death from intercurrent gastro-enteritis.

(1) In the brain. Arrested development of the cerebellum and of its efferent and afferent fibers, and of their nuclei of origin; of the restiform bodies, the olives, accessory olives, external arcuate fibers and nuclei arcuati, middle peduncles and transverse fibers of the pons, pontal nuclei, vestibulo-cerebellar tracts, superior peduncles, gray nuclei of cerebellum, red nuclei of tegmentum; possibly the cerebro-pontal tracts.

(2) In the cord. General attenuation of this structure throughout, division at the level of the fourth dorsal vertebra into two equal halves; marked enlargement of the central canal which divides with the division of the cord; the presence of various irregular cavities in the cord and disintegration of its central parts in the lower dorsal region; normal development of the gray matter with well-formed ganglion cells; scanty development of white matter, its place being taken by tissue of neuroglial structure.

(3) The coverings of the cord. The whole cord as far as the upper cervical area, embedded in a fine connective tissue with large vascular channels; the laminæ and spines of the fourth and fifth lumbar vertebræ deficient; an exostosis dividing the canal at the level of the fourth dorsal vertebra. In an appendix they give a complete bibliographical summary of cerebellar atrophies aranged on the basis of their proper pathogeny.

4. *Toxic Degeneration of the Lower Neurones.*—Dr. Barnes reports the histories of seven cases in which the clinical picture resembles in many respects that of peripheral neuritis and yet in other ways corresponds to the type of progressive muscular atrophy. The clinical histories are given very thoroughly, and his conclusions are very interesting. These may be summarized as follows: (1) There is a clinical type which is usually the sequel of acute specific fevers, which resembles the paralysis seen in multiple neuritis, but which is associated with great atrophy of the hand muscles. It usually begins about the second or third week after the febrile condition and involves the muscles from the periphery to the trunk to a varying extent. It may progress for a few days only, or for several months. Sensory signs are present, but slight in degree. Although the condition somewhat resembles progressive muscular atrophy, the prognosis and etiology of the two conditions are probably widely different from one another.

(2) After a certain stage, when once definite improvement has begun, relapses are not common, and there is a constant tendency to improvement. Even years after the subsidence of the acute condition considerable improvement may still take place, the small muscles of the hands being the last to recover. Contractures are rarely developed.

(3) Probably the condition is one of toxic degeneration of the lower neurones, the motor neurones being more particularly affected.

5. *Blood Changes in Idiopathic Epilepsy.* Dr. Robert Pugh of Claybury presents the results of the examination of forty cases of epilepsy in which differential counts of the leucocytes have been made, and the study of the alkalinity of the blood carried on. The author concludes that in epilepsy there are marked variations in the alkalinity of the blood, most of which manifest themselves by diminution of alkalinity. The average alkalinity during the (a) interparoxysmal period is lower than the average of the control cases. (b) There is a sudden pronounced fall immediately prior to the onset of the fit. (c) There is a certain diminution after the fit is over. Accounting for the interparoxysmal diminution the author suggests two hypotheses: (1) It may be explained by the gradual accumulation of toxines of an especial nature in the blood, and (2) that it may be the result of deficient metabolism, general metabolism being known to be below par in epilepsy. Accounting for the pronounced fall in the alkalinity immediately prior to the onset of the fit. The most likely origin of the acids the author claims are either (a) neuronic, (b) muscular. In the first case, the increased amount of acid being derived from the muscular and the glandular tissues. The author says that the exact rôle which the diminution in the alkalinity of the blood plays in the production of an epileptic attack is difficult to estimate. It is of utmost importance for any

cell in the body to have a proper adequate and pure supply of blood, and this applies especially to cerebral neurones, as they are extremely sensitive to a change in environment. The author believes that he has obtained good results in the treatment of epilepsy by maintaining the alkalinity of the blood within physiological limits, but unfortunately he says it is impossible to elevate and maintain the alkalinity by the administration of drugs.

With reference to the leucocytes, there is a distinct leucocytosis, particularly after the attack. The author's general conclusions are as follows: (1) The alkalinity of the blood in the interparoxysmal period is lower than the average of the control cases; (2) the diminution is gradual and progressive and is more marked in those cases suffering from gastric catarrh and constipation; (3) there is a marked sudden and pronounced fall immediately prior to the onset of the fit; (4) there is a further fall in the alkalinity after the fit is over; this diminution is seen from three to ten minutes after the attack; (5) this after-diminution depends upon the duration and severity of the muscular twitching, and upon the degree of the alkalinity in the interparoxysmal period; (6) there is a gradual return of the blood to its normal alkalinity, which takes place in five to six hours, the rise being more marked in the first hour.

JELLIFFE.

LE NEVRAXE

(Vol. IV. Fasc. 3. 1903.)

8. On the Form and Development of the Protoplasmic Prolongations of the Spinal Cells in the Higher Vertebrates. T. GEIER.
9. Researches on the Central Acoustic Tracts: The Bulbo Mesencephalic Acoustic Tracts. VAN GEHUCHTEN.
10. The Inhibitory Fibers of the Heart belong to the Pneumogastric Nerve and not the Spinal Nerve. VAN GEHUCHTEN.

8. *Form and Development of Protoplasmic Prolongations.*—Dr. Geier has been studying the dendrites of the spinal cells of the horn fibers, using kittens and rabbits for investigations. Golgi's method was used. He finds that the dendrites of the anterior horns are smooth in contour and differ from the posterior horns which have an irregular contour. The spinal dendrites extend in a straight line, while those in the posterior horn are irregular and sinuous. The dendrites in the cells of the anterior horns have few and insignificant collateral appendices while those of the posterior horns are quite complex. There are many variations in the dendrites of the new-born animal as contrasted with those of the adult, for full details of which the reader is referred to the original.

9. *The Upper Auditory Tract.*—Van Gehuchten here presents results of an extended series of experiments performed on rabbits and studied by the Marchi method. Five series of experiments are given which lead Van Gehuchten to different conclusions than those heretofore held. In the first group of cases he tore the seventh nerve out through the stylomastoid rim. In this way the ventral fibers of the trapezoid body were involved. Serial sections showed that degenerated fibers were found passing into the trapezoid body vertically above and below the actual level of the lesions. In the trapezoid these fibers could be traced to the opposite side; they turn upwards, those ventrad passing in front of and those dorsad behind the trapezoid nucleus. These two degenerated bundles so united to form a compact strand of fibers which lay in contact with the lateral aspect of the inferior nucleus of the lateral lemniscus. Tracing this strand of fibers upward it was found to turn outward and then backwards finally entering and forming the more superficial part of the lat-